MEDICAL STAFF CONFERENCE

Technique, Rationale, and Usefulness of Bedside Right Heart Catheterization In Critically Ill Cardiac Patients

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California, San Francisco. Taken from transcriptions, they are prepared by Drs. Martin J. Cline and Hibbard E. Williams, Associate Professors of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine.

DR. CRAWFORD:* This is the first Medical Center admission for this 61-year-old, white man, who was referred for evaluation of severe congestive heart failure following a myocardial infarction. The patient had been in good health until four years before admission, when hypertension was discovered on routine physical examination. He was treated with methyldopa and hydrochlorothiazide (Aldoril®). Eighteen days before admission, the patient experienced severe anterior, crushing chest pain radiating to the left arm. The pain was partially relieved by application of an ointment. Because of persistence of the pain the patient's physician had him admitted to hospital next morning.

Physical examination demonstrated evidence of mild, congestive heart failure, and an apical systolic ejection murmur was heard on auscultation of the chest. Electrocardiogram revealed evidence of an anterior septal myocardial infarction. On the 14th hospital day severe dyspnea suddenly developed. At that time an increase in the intensity of the systolic ejection murmur was noted and signs of congestive failure worsened. An electrocardiogram showed persistent ST segment elevation. The congestive heart failure could not be controlled with digoxin and diuretics and the patient was referred to the Medical Center.

On admission he was observed to be overweight, tachypneic, pale, diaphoretic. The pulse was 100 beats per minute; blood pressure, 100 mm of mercury systolic and 60 mm diastolic; respirations, 26 per minute; and temperature 38°C (100.4°F). The jugular venous pressure was elevated to the angle of the jaw at 45°, and the carotid pulsations were full. A flat percussion note was heard at the lung bases, with decreased breath sounds and rales above the flat percussion note area. Examination of the heart revealed a right ventricular heave, a systolic thrill at the left sternal border, a normal first heart sound, a wide but physiologically split second heart sound, and a left ventricular gallop. A 4/6 systolic ejection murmur was heard to be loudest at the left sternal border but could also be heard at the right sternal border and axilla. At the lower left sternal border the murmur seemed to increase with respiration. A three component friction rub was heard at the left sternal border. The liver edge was felt 8 cm below the right costal margin. The extremities were cool and pale, with good pulses and no edema.

The patient was treated vigorously with digoxin, diuretics, and fluid restriction until multiple uni-

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focal, ventricular premature contractions developed. He was then transferred to the coronary care unit and digitalis was withheld. Several days later, results of bedside right heart catheterization, performed by means of a small, flow-directed catheter, were diagnostic of ventricular septal defect. This diagnosis was confirmed by preoperative angiography. The patient's condition was stabilized on a vigorous medical management until the 69th day following myocardial infarction. On this day left ventriculotomy was carried out, with excision of a large aneurysm of the anterior left ventricular wall and repair of a 3 cm ventricular septal defect. The postoperative course has been uncomplicated and the patient is feeling quite well.

DR. SMITH:* Thank you very much, Dr. Crawford.

Dr. Gold, may we see the radiographic films?

DR. GOLD:† The initial chest radiograph taken at the time of hospital admission demonstrated prominent pulmonary veins, suggesting moderate congestive heart failure with cardiomegaly and bilateral pleural effusions. Several days later signs of congestive heart failure appeared to be increased almost to the point of pulmonary edema. On later radiographs very prominent pulmonary arterial vessels appeared which looked like shunt vessels related to the presence of the interventricular septal defect.

DR. SMITH: The patient is not actually here for personal presentation this morning, although I gather he is doing quite well. We have asked Dr. Melvin Scheinman and Dr. Joseph Abbott to discuss this patient and the diagnostic approach of bedside right heart catheterization.

Discussion by Dr. Scheinman‡ and Dr. Abbott§:

Rupture of the intraventricular septum is a rare and frequently fatal complication of acute myocardial infarction.[1,2] This complication is most likely to occur within the first two weeks when the area of infarction is softest. The diagnosis is suggested by the abrupt appearance of signs and symptoms of both left and right heart failure together with a loud holosystolic murmur in a patient with a recent myocardial infarction. The electrocardiogram usually shows evidence of a recent transmural myocardial infarction and may reveal intraventricular conduction delays. The chest roentgenogram is characterized by an enlarged cardiac silhouette and pulmonary vascular congestion. Approximately 25 percent of these patients die within the first 24 hours and over 81 percent succumb within eight weeks.[2] Postmortem examination shows extensive infarction of both the muscular septum and the free wall of the left ventricle.

The clinical presentation of postinfarction ventricular septal defect often resembles that of acute mitral insufficiency resulting from rupture of a papillary muscle.[3] Correct diagnosis is more than an academic exercise because successful repair of septal perforations is possible.[4] The case presented this morning aptly demonstrates the value of bedside right heart catheterization for both diagnosis and quantitation of an acquired left-to-right ventricular shunt. In addition, the technique is of great value in monitoring critically ill cardiac patients.

Commonly used methods of hemodynamic monitoring are designed to determine whether left ventricular filling pressure and systemic flow are adequate to meet tissue needs. Left ventricular filling pressure can best be determined by direct measurement of left ventricular end-diastolic pressure (LVEDP). This can be carried out at the bedside by percutaneous insertion of a stiff, relatively large-bore catheter into a peripheral artery with retrograde passage into the left ventricle.[5,6,7] A recent collaborative study[8] documented the incidence of various complications (arterial thrombosis, hemorrhage, arrhythmia, and ventricular perforation) in patients undergoing left heart study in a cardiac catheterization laboratory. One can reasonably predict a significantly higher incidence of complications and death in critically ill patients studied at the bedside. Measurement of central venous pressure (CVP) is a more popular method of assessing left ventricular filling pressure.[9,10] Our studies, together with those of Loeb and coworkers,[5] show that CVP may in fact be a misleading index of LVEDP, especially in patients with acute myocardial infarction treated with inotropic or vasopressor agents.

Therefore, we use an alternative method of assessing both left ventricular filling pressure and systemic flow — namely, bedside flow-directed catheterization of the pulmonary artery. One end

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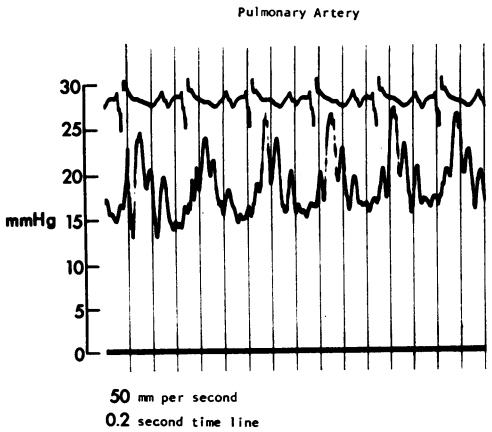

Chart 1.—A phasic tracing of the pulmonary arterial pressure wave recorded from the miniature catheter. The simultaneously registered electrocardiogram (upper tracing) allows correlation of electrical and mechanical events.

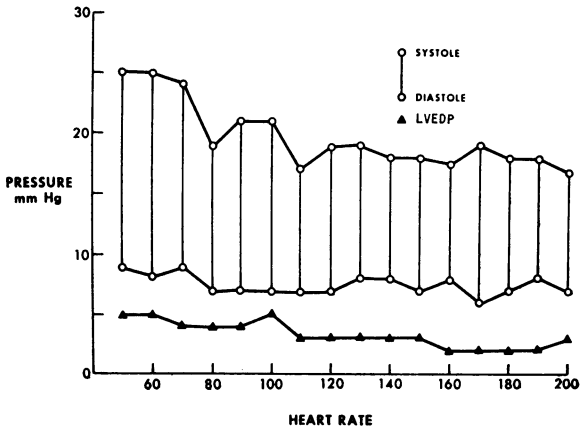

Chart 2.—The effect of change in heart rate on pulmonary artery diastolic and left ventricular end-diastolic pressures. The values represent averages of the control state in five canine experiments wherein heart rate was varied by atrial or ventricular pacing after sino-atrial heart block was induced. No increase in pulmonary artery diastolic pressure was produced by the tachycardia. Within the range of the heart rates studied, the pulmonary artery diastolic pressure accurately mirrored left ventricular end-diastolic pressure. (LVEDP=left ventricular end-diastolic pressure.)

of a 100 cm-long nylon catheter* (outside diameter, 0.37 inch; wall thickness, 0.07 inch) is flared to fit an adapter. The adapter is connected to a pressure transducer, and permanent records are obtained by recording simultaneously both the pressure waves and the electrocardiogram. The catheter, connected to the transducer, has a resonant frequency of 40 cycles per second with a damping coefficient of 0.40 at 98.6°F (37°C). Underdamped phasic pressure waves are recorded and are comparable in contour to those obtained from the larger and stiffer catheters used in the cardiac diagnostic laboratory (Chart 1). No premedication is required. The right or left antecubital fossa is scrubbed with hexachlorophene (pHisoHex®) and thimerosal (Merthiolate®) and draped appropriately. An 18-gauge, thin-walled needle is inserted into a medial antecubital vein, and the catheter is advanced through the needle into the vein. The catheter is flushed with a dilute heparin sodium solution and gently advanced into the pulmonary artery under electrocardiographic and pressure monitoring. Fluoroscopy is not employed. Abduction or external rotation of the limb usually overcomes any resistance to passage at the axilla. When the catheter is in the right atrium (judged by the length of catheter passed and the contour of the pressure record), introduction into the right ventricle and pulmonary artery is often facilitated by having the patient inspire deeply or rotate to either a right or left lateral decubitus position.

Pulmonary artery pressure is determined by the pulmonary blood flow, the resistance and compliance of the pulmonary vascular bed, and the level of intra-alveolar, intrathoracic, and left atrial pressures.[11] In general, for any given left atrial pressure, changes in pulmonary artery systolic pressure reflect changes in stroke volume, while changes in diastolic pressure are indicative of changes in pulmonary vascular resistance.[12] Normally, the pulmonary capacitance is so great and its resistance so low that pulmonary artery diastolic pressure would be expected to be equal to or slightly greater than the pulmonary venous, left atrial or left ventricular pressures at diastasis.

Recently we studied the relationship between pulmonary artery and LVED pressures over a wide range of heart rates and left ventricular filling pressures in the open-chest, anesthetized dog. Catheters were inserted into the pulmonary artery, the left atrium, and the left ventricle while heart rate was controlled by sino-atrial node block and atrial or ventricular pacing. Using this preparation, heart rate was varied from 50 to 200 beats per minute. At the slowest heart rates, the difference between diastolic pulmonary and left ventricular pressures varied from 0 to 5 mm of mercury and graded increases in heart rate produced little change in the diastolic gradient (Chart 2). Similarly, when LVEDP was raised, either by increasing afterload (by mechanical aortic constriction or after administration of methoxamine hydrochloride) or preload (by rapid infusion of saline solution), both

*Portex®, Smith Industries, Ltd., Jamaica, New York.

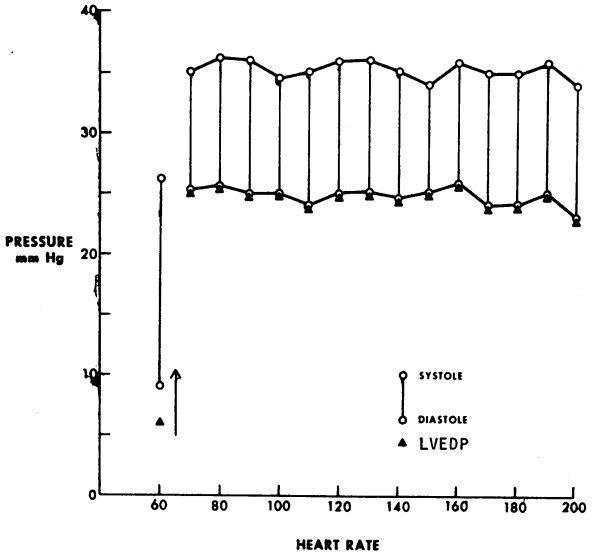

Chart 3.—The effect of change in heart rate on pulmonary artery diastolic and left ventricular end-diastolic pressures after increases in afterload or preload had induced an elevated left ventricular end-diastolic pressure (arrow). The values represent averages of five canine experiments wherein heart rate could be controlled by sinoatrial block and cardiac pacing. The gradient between the pulmonary artery diastolic and left ventricular end-diastolic pressures is abolished by elevation in left ventricular pressure. The identity between the diastolic pressures is maintained from the slowest to the fastest rate. (LVEDP= left ventricular end-diastolic pressure.)

diastolic pressures were identical over a wide range of heart rates (Chart 3). Thus, although the diastolic period was significantly shortened by acceleration of heart rate, the pulmonary artery diastolic pressure remained a sensitive indicator of LVEDP and this relationship was constant over a wide range of left ventricular filling pressures (0 to 25 mm of mercury).

Similar results have been reported in normal subjects studied over a wide range of heart rates and levels of cardiac output.[13] Kaltman and coworkers[14] showed a close correspondence between pulmonary artery and LVED pressures in patients with acquired heart disease: LVEDP varied from 6 to 18 mm of mercury and heart rate ranged from 60 to 140 beats per minute. Thus, pulmonary artery diastolic pressure appears to be a reasonable index of left ventricular filling pressures over a wide range of heart rates and ventricular end-diastolic pressures in both dog and man.

Certain limitations in the use of pulmonary artery diastolic pressure as a measure of LVEDP are apparent. First, patients with obstruction to pulmonary flow (either at the levels of pulmonary artery, capillary, or vein, or of left atrium or mitral valve orifice) have a gradient of pressure at the end of diastole. Although pulmonary artery diastolic pressure is an invalid measure of the absolute level of LVEDP in these patients, changes in pulmonary artery diastolic pressure probably reflect changes in LVEDP. Patients with pulmonary hypertension secondary to chronic obstructive bronchopulmonary disease, for example, show parallel rises in pulmonary artery diastolic and pulmonary capillary wedge pressures following rapid intravenous infusions of dextran.[15] In addition, acidosis and hypoxia have important effects on pulmonary artery diastolic pressure independent of left ventricular filling pressure and therefore must be corrected before estimations of left ventricular competence are made on the basis of pulmonary artery diastolic pressure.[12] Finally, LVEDP can actually exceed pulmonary artery diastolic pressure in patients with congestive heart failure or with inflow obstruction secondary to severe left ventricular hypertrophy.[16,17] Augmentation of the atrial contraction results in a large "a" wave in the left ventricular pressure pulse with consequent elevation of the LVEDP. The "reversed" gradient is small in magnitude and probably of little clinical significance.

Our own experience has shown the pulmonary artery diastolic pressure to be a more sensitive index of LVEDP than CVP. For example, one-third of our patients with acute myocardial infarction complicated by pulmonary edema had a normal CVP at a time when pulmonary diastolic pressure was decidedly elevated.[18] A more significant finding was the poor correlation between changes in CVP and changes in pulmonary artery diastolic pressure.[19] Thus, the CVP often is an inaccurate reflection of left ventricular filling pressure.

We initially emphasized the desirability of monitoring systemic flow in critically ill patients. However, it is apparent that the critical factor in determining survival of patients in shock, for example, is not the level of systemic pressure *per se,* but rather the adequacy of the cardiac output to meet tissue needs. Goldman and coworkers[20,21] recently described the clinical usefulness of measurements of central venous oxygen saturation (CVO_2) in monitoring patients with acute myocardial infarction. They found that patients with CVO_2 saturations of 60 percent or less tend to show evidence of either heart failure or shock or a combination of the two. This finding is not surprising since diminished cardiac output would be expected

to result in greater extraction of oxygen by peripheral tissues and therefore an abnormally low oxygen saturation in blood returning to the heart. Furthermore, it is apparent from the Fick equation

$$\text{(cardiac output} = \frac{\text{oxygen consumption}}{\text{arterial-mixed venous oxygen content}})$$

that cardiac output varies directly with mixed venous oxygen content when the oxygen consumption and arterial oxygen content remain constant. Although the CVO_2 tends to be slightly lower than the mixed venous oxygen saturation (MVO_2)[22] (because of the large contribution of highly-saturated renal venous effluent returning via the inferior vena cava[23]), the CVO_2 is still a reasonably valid reflection of MVO_2 in normal subjects or in seriously ill patients without evidence of heart failure or shock. However, in patients in cardiogenic shock we found a reversal of the normal relationship in that CVO_2 was consistently higher than MVO_2.[24] These findings are compatible with the thesis that low output states are attended by redistribution of blood flow away from the splanchnic, renal, and mesenteric beds toward the cerebral circulation, accounting for the disparity between CVO_2 and MVO_2. These results do not negate the usefulness of serial CVO_2 measurements in patients with severe low output states, since changes in CVO_2 are far more important than the absolute levels and correlate well with corresponding changes in MVO_2 in these patients.[24]

In addition to measuring right heart pressures and MVO_2, the flow-directed catheter technique allows for measurement of cardiac output by the Fick method when arterial blood and expired air are collected. Blood samples can also be obtained from chambers in the right side of the heart to establish or eliminate the presence of a significant left-to-right shunt. In addition, catheterization of the pulmonary artery allows quantitation of right-to-left shunts.[25] Depending on the clinical indications, the catheter may be left in place for hours or days. In this instance, the introducing needle is withdrawn from the vein and the catheter is taped directly to the skin and kept patent by a continuous dilute heparin drip.

In the past two and a half years we have attempted catheterization of the right heart at the bedside in 114 patients and succeeded in entering the pulmonary artery in 96 patients and only the right ventricle in 18 patients. The time necessary for completion of the procedure varied, but most studies were performed within 30 minutes. There were no instances of lost or knotted catheters, phlebitis, emboli, infection, or death. Ventricular premature beats occurred commonly as the catheter entered the right ventricle, but in only three instances did a serious arrhythmia occur: Ventricular tachycardia developed in two patients and transient atrial fibrillation in the third patient. Ventricular tachycardia stopped spontaneously on removal of the catheter in one patient, although the other patient required a single 200 joule direct current countershock to the chest. These instances were unusual, however, for the catheter was well tolerated by most patients and has been left in the pulmonary artery for as long as six days.

The procedure is safe, provided certain precautions are followed:

- Catheterization should not be performed during the early phases of acute myocardial infarction, especially if the patient shows pronounced ventricular irritability. The two catheter-induced ventricular arrhythmias occurred in patients with acute myocardial infarction and premature ventricular beats who were studied within 12 hours of hospital admission.
- Patients with ventricular irritability should be treated with the appropriate antiarrhythmic agents before catheterization. The procedure should then be performed in an intensive care area with access to lidocaine and a direct current defibrillator. Since the establishment of these precautions, no serious arrhythmia has occurred in the patients studied.

In summary: The procedure is safe and easily mastered; in fact, most studies were performed by house officers under the supervision of the authors. Use of the pulmonary artery diastolic pressure as a measure of left ventricular filling pressure is valid in principle. In addition, the technique allows for serial measurements of MVO_2 saturation or cardiac output and is thus extremely valuable in assessing hemodynamic dysfunction and the response to various therapeutic interventions in critically ill patients.

DR. SMITH: Before discussing these interesting suggestions and this technique, we should call on Dr. Benson Roe to comment more specifically about this patient and the findings at operation.

DR. ROE:* I would like to stress the immense value derived from the routine use of the left

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atrial catheter in postoperative management of the valve replacement patient with low output syndrome. The inability to monitor the left side of the heart is a serious handicap in the management of left-sided functional abnormalities. We feel that the surgical opportunities in the treatment of patients with these severe consequences of acute myocardial infarction have not really been adequately utilized. All patients who are in shock or with very low cardiac output as a result of myocardial infarction should be monitored by this modality, their hemodynamic abnormality carefully assessed, and the opportunity for surgical intervention properly planned.

We felt it desirable to manage this patient conservatively so long as possible in the hope that the surgical techniques could be facilitated by the availability of scar tissue rather than dead myocardium. At a surgical meeting last week, the group from the Chicago Presbyterian Hospital reported six patients operated on following acute decompensation after myocardial infarction with five survivors. Three of these patients were operated on within two and a half weeks of their initial infarction and all survived. I feel that today's patient demonstrates an opportunity which is perhaps being neglected frequently in patients with severe myocardial infarction.

DR. SOKOLOW:* I would like to reemphasize the importance of doing bedside cardiac catheterization in a patient of this sort, particularly in view of the difficulty in delineating a ruptured ventricular septum from mitral insufficiency secondary to malfunction of the papillary muscle. The murmurs are identical and cardiac failure may develop in both situations. Differentiation is important because mitral insufficiency can be corrected early in the course of the disease, whereas a ventricular septal defect requires ideally a period of one to three months before the repair can be made. In this patient the important factor in making the diagnosis was the oxygen content of the right ventricle. When failure develops in patients with myocardial infarction, one should employ these newer techniques in order to establish the diagnosis so that surgical treatment can be instituted. I think everyone considered the possibility of ruptured ventricular septum in this patient, but differentiation from mitral insufficiency could not be made until the catheter data became available.

*Maurice Sokolow, M.D., Professor of Medicine.

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